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Animal and Plant Health Inspection Service

Program Aid 1578

Keeping America Free From Foreign Animal Diseases

Contagious Bovine Pleuropneumonia

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Volume 3

Guidelines for Using This Package

This binder contains an integrated suite of educational materials about contagious bovine pleuropneumonia. The package can be used in a formal training setting, where a presenter will show the video tape and narrate the slide show using this black-and-white brochure as the script. Or the materials can be used in a self-study program with the reader progressing at his or her own pace.

Within this brochure, readers will notice that certain paragraphs are preceded by a number. These numbers correlate to the slide set. For example, the contagious bovine pleuropneumonia slides are all marked "CBPP" at the top of each plastic slide frame and numbered sequentially from 1 to 38.

If you remove the slides from their protective clear-plastic sleeve (for example, to put them into a carousel for group viewing), please be sure to reposition them in the correct numeric order for the benefit of future users.

This shrink-wrapped suite includes a general and a scientific video tape on contagious bovine pleuropneumonia, a slide set on the disease, and the brochure you are reading now. If your package is incomplete, please contact the following office for replacement materials:

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Veterinary Services, Emergency Programs
4700 River Road, Unit 41
Riverdale, MD 20737–1231

Instructional packages on other diseases are also available and may be requested by writing to the above address. Titles include
Program Aid 1576 African Horse Sickness
Program Aid 1577 African Swine Fever
Program Aid 1579 Lumpy Skin Disease, Sheep Pox, Goat Pox
Program Aid 1580 Malignant Catarrhal Fever
Program Aid 1581 Rinderpest, Peste des Petits Ruminants
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Contagious Bovine Pleuropneumonia

Definition

1 Contagious bovine pleuropneumonia (CBPP) is a highly contagious acute, subacute, or chronic infection primarily of cattle caused by *Mycoplasma mycoides* subsp. *mycoides* (small-colony type, abbreviated "SC" hereafter). The disease is characterized by sero-fibrinous pleuritis, pulmonary interlobular septa thickened by edema and/or connective tissue, and consolidation and necrosis in the lung.

Etiology

2 The causative organism is 125 to 175 μm in size, is pleomorphic, and can be cultivated in cell-free media.

The organism is sensitive to drying and heating and does not survive for more than a few days when exposed to normal environmental conditions. It also does not survive in meat or meat products.

M. mycoides subsp. *mycoides* (SC) is culturally and antigenically similar to *M. mycoides* subsp. *mycoides* (large-colony type). In some parts of the United States, the latter organism causes polyarthritis, pleuritis, and pneumonia in goats but is not known to infect cattle.

Effective Disinfectants

M. mycoides is readily susceptible to most routinely used disinfectants.

History

3 The first record of CBPP was in Germany in 1693. From Germany, the disease spread over all Europe. In 1843, it was introduced into the United States via a dairy cow that was purchased off a ship from England. By 1884, CBPP had become so widespread and destructive in the United States that the Federal Government established the Bureau of Animal Industry to combat the disease. In 1887, the first intensive campaign to control an animal disease by quarantine and slaughter began. The disease was eradicated from the United States in 1893.

M. mycoides subsp. *mycoides* (SC) was first isolated and cultivated in artificial media in France by Nocard and Roux in 1898.

Illustrating how quickly CBPP can spread, in about 1854, CBPP was introduced into South Africa by a Freisland bull imported from Holland. The disease spread rapidly and in 2 years caused the death of 100,000 cattle. The disease was eradicated from South Africa in 1916.

CBPP was eradicated from Australia in 1971.

Host Range

4

CBPP is a disease primarily of cattle—both bovine and Zebu cattle. Rarely, infections have been reported in other species, mostly from zoos—in yak, bison, reindeer, and antelopes. Water buffalo is susceptible, but natural cases are rare. In a serological survey of wild wildebeest, impala, eland, topi, waterbuck, African buffalo, hippopotamus, and zebra, all animals tested negative.

Strains of *M. mycoides* subsp. *mycoides* (SC) have been adapted to mice, rabbits, guinea pigs, and hamsters.

Geographic Distribution

5

CBPP is present in parts of Africa, Asia, Spain, and Portugal.

Transmission and Epidemiology

6

CBPP has to be introduced by an infected animal. The disease is spread primarily by inhalation of droplets produced by an infected animal's coughing. Close contact, particularly housing of animals, is required for transmission. In some animals, the disease can be very mild: the animal will appear normal but may be shedding the organism for months or years before being recognized as infected.

M. mycoides subsp. *mycoides* (SC) has been recovered from the urine of severely infected animals, and the disease was transmitted experimentally to other animals by feeding hay contaminated with urine-diluted broth cultures.

Animals are able to wall-off (sequester) an area of infected lung. Traditionally, it has been believed that when an animal with a sequestrum was stressed—exhausted, starved, or affected by intercurrent disease—the capsule would break down and the organisms would be shed. Recent studies in Africa suggest that, in CBPP, this method of spread may not be important.

Initially, CBPP was difficult to reproduce experimentally. The first experimental transmission was by intrajugular injection of cultures mixed with 10-percent agar. Today animals are experimentally inoculated by intrabronchial intubation.

Incubation Period

7

Under natural conditions, the incubation period ranges from 10 days to 6 months. The mean incubation period in contact animals is 3 to 6 weeks. The rate of spread and incubation period depend on the method of husbandry.

In experimental animals inoculated by intrabronchial intubation, the incubation period is usually 2 to 3 weeks.

Pathogenesis

8

M. mycoides subsp. *mycoides* (SC) apparently produces a potent toxin, for if the organism is inoculated intramuscularly or subcutaneously, a large area of edema and necrosis quickly develops. This phenomenon can also be seen in the lung. Initially, there will be a small focus of alveoli filled with inflammatory cells; the area will undergo coagulative necrosis, and the severity of edema will decrease as the distance from the necrosis increases (dilution by diffusion). The edema is best seen as distention of interlobular septa. If the animal cannot control the infection, the area rapidly enlarges and reaches the visceral pleura, and abundant fluid and fibrinous exudate accumulate in the thoracic cavity. If the animal controls the infection, a lymphocytic infiltration into the periphery of the distended interlobular septa is followed by fibroblasts. Eventually, the interlobular septa become thick, white bands of connective tissue. While this process is advancing, a connective-tissue capsule develops around the necrotic area of lung to form a sequestrum. One distinguishing feature of CBPP lesions is coagulative

necrosis rather than the liquefaction necrosis seen in many other pneumonias in cattle.

In some severe cases of CBPP, the animals will develop septicemia, and the CBPP agent will localize and grow in joints. Affected joints become very enlarged by proliferated connective tissue and exudate. This form of the disease is seen mostly in calves.

Clinical Signs

9

Acute cases of CBPP have signs typical of pneumonia and pleuritis:

- Fever 105 °F (40.5 °C)
- Anorexia
- Depression
- Tendency to stand away from other animals
- Reluctance to move
- Painful, difficult breathing
- Rapid shallow breathing
- Cough
- Head lowered and extended, elbows out

(If the animal is forced to move quickly, breathing becomes more distressed and the animal may cough.)

About half of the animals die in the acute state of CBPP. Death occurs in several days to 3 weeks after the onset of signs. Animals that are going to die lose condition and develop labored breathing accompanied by grunting. Young animals may develop enlarged joints.

10 **Chronic disease** may develop in animals that do not die. These animals are unthrifty, have recurring low-grade fever, and shed the causative agent.

Animals with **less acute cases** have signs of pneumonia for 3 to 4 weeks and then make an apparent recovery, but the infection persists and the animal may shed the organism.

A small number of animals infected with *M. mycoides* subsp. *mycoides* (SC) do not become febrile or show any sign of infection but are capable of transmitting the disease.

11 A coughing animal.

12 An emaciated animal.

Gross Lesions

13 In the typical acute case of CBPP, usually only one lung is involved. If there are lesions in both lungs, the lesions are not symmetrical. This distribution is important for distinguishing CBPP from other pneumonias. The thoracic cavity on the affected side may contain a gallon or more of clear yellow or turbid yellow fluid mixed with strands of fibrin. The infected area of lung is usually adhered by fibrin and connective tissue to the thoracic wall. The amount of connective tissue is an indication of the age of the lesion. If the lung appears normal, the best way to find a small lesion is to do a thorough palpation.

The affected areas of lung are firm and raised above the normal adjacent lung. When the firm area is cut, it is odorless and has the typical marbled appearance of CBPP—red, grey, or yellow lobules outlined by yellowish (edematous) or white (connective tissue) interlobular septa and subpleural tissue. The distension of the interlobular septa decreases as the distance increases from the center of the lesion. Necrotic areas of lung have an opaque appearance, and normal lung structure is still evident—coagulative necrosis. These necrotic areas may be 6 to 7 cm in diameter or larger and may be surrounded by connective tissue—sequestrum.

Small CBPP lesions will have the same recognizable appearance as large lesions.

Occasionally, the sequestered area will undergo liquefaction necrosis; this probably results from bacterial replication in the sequestrum and the attraction of neutrophils.

In more resistant animals or animals infected by a less- virulent strain, interlobular septa around the site of the lesion will be widened by connective tissue. The lobules may be normal or consolidated.

Affected joints are enlarged. When they are opened, the general area displays a great increase in connective tissue in the joint capsule, a large amount of turbid synovial fluid, and fibrin in the joint.

Photographs of Gross Lesions

14 Large mass in the left lung. A lesion in only one lung is typical in CBPP.

15 A closeup from slide 14.

16 Both lungs are distended. The left lung is distended by air; the right lung has a large consolidated area in the diaphragmatic lobe, and there is fibrin over the surface of the lung.

17 A large amount of fibrin on the pleural surface of the ribcage.

18 Section of lung has the typical marbled appearance associated with CBPP. The white areas are interlobular septa thickened by connective tissue. The lobules in the area are consolidated or ateliotic.

19 A closeup view of a similar area.

20 A section from another lung. Part of the lobe has very thickened interlobular septa; the lobules in the pink area in the central lower part of the photograph contain air and are separated by widened (edematous) interlobular septa.

21 A section from another lung in which the interlobular septa are widened by connective tissue. The presence of connective tissue means the lesion has been present for a while.

22 Closeup of a similar area.

23 In this section of lung, the subpleural area is thickened. In the rest of the section, the lung structure can be seen, but the lung has an opaque appearance; the entire lung has undergone a coagulative necrosis.

24 The joint is enlarged due to a proliferation of connective tissue.

25 The tendon sheath had an increase in fluid.

26 The fluid in the joint is increased in amount and turbid.

Microscopic Lesions

In the early lesion, a few alveoli are filled with inflammatory cells, and adjacent interlobular septa are edematous. As the lesion becomes larger, the center undergoes coagulative necrosis, and fibrin is deposited around the necrotic area. If the animal controls the infection, the area is walled off and there is lymphocytic hyperplasia around adjacent blood vessels and bronchi; otherwise, the lesion continues to enlarge.

The interlobular septa around the affected lobule initially become distended with fluid. Then a lymphocytic infiltrate starts at the sides of the septa and moves toward the center; this process is followed in the same way by fibroblasts.

Photomicrographs

27 Alveoli filled with fluid and a few inflammatory cells.

28 Higher magnification of slide 27.

29 Pneumonia with an area of necrosis in the center of the photograph.

30 Large area of coagulative necrosis.

31 Greatly distended lymph vessel in an edematous interlobular septum; there is a small amount of lymphocytic infiltrate. This is an early stage in the development of the connective-tissue-widened interlobular septum.

32 A later stage in the development of a connective-tissue- widened septum. At this stage, there is an increase in fibrin and cells in the septum and a dense lymphocytic infiltrate at the margin of the septum.

Morbidity and Mortality

33 Morbidity associated with CBPP is variable, in part dependent on husbandry. Confinement increases morbidity. Mortality can range from 10 percent to 70 percent.

Diagnosis

For CBPP as for other diseases, the initial case in a free area has to be confirmed by isolation and identification of the causative organism.

Field Diagnosis

34 The clinical signs of CBPP are indistinguishable from other pneumonias. Observation of a lesion typical of CBPP should make one suspect the disease.

35 **Specimens for Laboratory**

- Piece of the lung lesion. Section should include necrotic and viable tissue.
- Pleural fluid
- Bronchial lymph node
- Joint fluid

Preferably, specimens should be placed in a transport medium—heart infusion broth, 20-percent serum, 10-percent yeast extract, benzylpenicillin at 250 to 1,000 IU per mL—and be shipped on wet ice. If transport to the laboratory will take more than a few days, specimens may be frozen.

Laboratory Diagnosis

36 **Isolation**—The organism can be isolated on specialized mycoplasma medium. After isolation, the organism is identified by biochemical tests, slide agglutination test and/or immunofluorescent test.

Serology—Antibody to CBPP can be detected using a complement-fixation (CF) or enzyme-linked immunosorbent assay (ELISA).

The CF test will detect 100 percent of acute cases and about 70 percent of chronically affected animals.

Vaccination

37 The first CBPP vaccine was a partially attenuated *M. mycoides* subsp. *mycoides* (SC) produced in a calf. The vaccine was lymph collected from a site of subcutaneous inoculation. The lymph was inoculated into the tip of the tail or end of the nose. A localized infection (and sometimes necrosis) would develop and induce an antibody response.

Later, an egg-adapted attenuated vaccine was produced.

The CBPP vaccine used today is an avianized vaccine grown in broth culture. In Africa, this vaccine is frequently combined with rinderpest vaccine.

Control or Eradication

38 Because CBPP may exist as a subclinical or chronic disease, it is important to prevent its introduction by restricting importation from infected areas and performing serologic testing of imported animals.

When CBPP is diagnosed in a previously uninfected area, all infected animals should be slaughtered, buried, or incinerated, and all exposed animals should be strictly quarantined, observed, and tested serologically for a year.

In areas where CBPP is enzootic, cattle should be vaccinated annually.